which the rectus muscle was markedly rigid. By 9 A.M. on September 7, when I first saw the patient with his family doctor, Dr. George Cameron, his condition was as above described, only the shock was more intense and the pain unrelieved. His entire abdomen was tender, but was more intense in the left hypochondriac region. The mass in this locality was readily mapped out in spite of the muscular rigidity. The pneumothorax had increased until the apex beat of the heart was to the right of the sternum. Breathing was labored, and he experienced considerable difficulty on inspiration.

Surgery being out of the question a medical consultant was advised, and Dr. George M. Piersol was asked to see the case. He arrived about 11.15 A.M., and while making an examination the patient expired.

In the absence of tuberculosis and traumatism, except the spell of vomiting, and of cough or bloody expectoration, and with the history of prolonged stomach dyspepsia with pain, a diagnosis of perforated gastric ulcer at or near the cardiac end of the stomach was made. The explanation of the pneumothorax was that the ulcer was adherent to the under side of the diaphragm, and when perforation occurred it included the diaphragm and parietal layer of the pleura.

A postmortem was refused, so that this diagnosis must remain unconfirmed and a matter of speculation. However, the evidence seems sufficient to justify the inclusion of the case in this series.

REPORT OF A CASE OF PAROXYSMAL TACHYCARDIA WITH OBSERVATIONS ON TREATMENT.

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The patient, a married man, aged thirty-seven years, has continuously been under my observation since March 18, 1909. Cardiac valvular disease was first recognized when he was eighteen years of age, and, according to the family legend, he "strained" his heart at about that time, while rowing. Careful questioning fails to elicit a history of any illness which might be interpreted as acute rheumatic fever or any other infection which would be likely to involve the heart. He was always extremely moderate in the use of alcohol and tobacco, and venereal infections were denied.

The first paroxysm of tachycardia came on in 1905, immediately after "shooting-the-chutes," and during the next six years he experienced many repetitions. The attacks varied in duration from a minute or two to twenty-eight hours, the average being from four to six hours. Their frequency was also variable, sometimes recurring three or four times a day, while at other times there were intervals of freedom lasting several weeks; the longest was nine weeks. These attacks did not appear to be precipitated by any specific cause, although much exertion would quite surely cause one, as would, apparently, any toxemia.

In 1907 he is said to have had a mild attack of "grip," which was followed by jaundice lasting one week, during which time he had a great number of attacks.

I have frequently noted that a mild coryza, or tracheitis, was likely to be accompanied by paroxysms, and that stasis in the intestinal tract would quite surely precipitate them, often being preceded by a few urticarial spots. I attempted to determine whether any specific protein caused them, but was unable to arrive at such a conclusion. They quite often started at night, and the patient thought they were more apt to occur if he lay on his left side when sleeping, or they began while working at his office, or while eating. He was always immediately aware of the onset of an attack by a sensation of fulness in his chest and throbbing of his heart. He usually went to bed—if not already there—and sat upright with pillows at his back. He had no dyspnea except in long attacks, but was distinctly uncomfortable if he tried to lie down. After about an hour he would complain of "feeling sick all over," having pain in his head, back, and limbs, and constant nausea with frequent vomiting of considerable mucus. He constantly had a mild degree of icterus, and during long attacks this became deeper.

The attacks always ceased abruptly with a "bump" of his heart, and the pulse would then immediately become slow and regular. Within a few minutes he would feel quite well, and could shortly take food. There were no premonitory signs to indicate either the onset or offset of attacks.

Examination between paroxysms showed a man of spare build and poorly developed muscles, with a faint icterus in the skin and scleræ. The pulse was 72 to 80 per minute, regular, and of normal size and force. The vessels were not palpable. Systolic blood-pressure, 110; diastolic, 75. The heart transmitted a diffuse, heaving impulse to the precordium, the point of maximum impulse being in the sixth left interspace, 11 cm. from the median line. The apex beat was in the sixth space, 13.5 cm. to the left of the median line. There was no undue pulsation of the cervical vessels. The upper border of cardiac dulness began at the second rib, the left border was 14.5 cm. to the left and the right border 5 cm. to the right of the median line. Auscultation revealed a long, low, harsh systolic murmur, best heard at the maximum point of impulse, which could be traced into the left axilla and below the angle of the left scapula, also over the body of the heart and 3 cm. to the right of the midsternal line in the fifth interspace. A long, rumbling, crescendo diastolic murmur, which ceased abruptly with a sharp, loud shock to the first sound, was also heard over a small area just to the right of the apex beat. The pulmonary second sound was much accentuated. Polygraphic tracings showed normal jugular and radial curves. The lungs and abdominal viscera were negative. There was no ascites or edema, and the urine and blood were normal. Wassermann reaction negative.

During attacks the patient was usually found sitting up in bed, looking sick and anxious, but he was not cyanotic. The whole left side of the thorax would be rapidly heaving, and a wavy impulse was seen in the fourth, fifth, and sixth interspaces. The most striking feature of inspection was, however, exceedingly violent throbbing of the jugular bulbs, which was so great that it was impossible to get tracings directly from them. The systolic blood-pressure was 90 to 95. Except in very long attacks, dilatation of the heart could not be demonstrated. The heart sounds, which were perfectly regular, varied from 140 to 190 per minute, usually being about 170. A short, faint, systolic murmur could be heard over the precordium. The pulse was very small, but there was no deficit.

Since the onset of attacks of tachycardia the patient has led a very quiet life, being largely freed from business cares. A great variety of therapeutic measures for stopping the paroxysms were tried, but none, except that noted later, seemed to be of value if the paroxysms lasted more than an hour. They sometimes ceased, with vomiting or defecation. Pressure on the vagi, changes in position, and prolonged holding the breath were without avail, except the latter, which frequently terminated short attacks and was most relied upon by the patient.

It was difficult to get tracings from the jugular bulbs during paroxysms because of their tremendous throbbing, but those obtained show the ventricular form of the venous pulse (Fig. 1). It is quite certain that the auricle was not fibrillating as the heart was always regular, and Lewis has concluded "That auricular fibrillation is never accompanied by regularity of the ventricle except when it is complicated by auriculoventricular heart-block, and it appears reasonable to assume that when the ventricular form of venous pulse is present and the pulse is regular and of normal or increased rate the auricle is not in a state of fibrillation."

During the fall of 1911 the patient was about as usual, having short attacks every few days until November 13, when he had a paroxysm lasting twelve hours. It was accompanied by no unusual features, and on November 14, although he remained in bed, he felt perfectly well and reported his heart to have been slow and regular all day. However, at 7 P.M. it became rapid (140 to 180 per minute), and so continued, except for short intervals subsesequently noted, until 8.30 P.M., November 21, a period of one week. Within forty-eight hours it became extremely dilated. There was congestion of the lungs, with expectoration of bloody sputum, the liver became large, tender, and pulsating, and there was deep icterus and edema of the lower extremities. During the first twenty-four hours he was constantly nauseated, but this subsided later. He felt weak and faint, and much of the time was stuporous, with Cheyne-Stokes respiration. All of the usual measures were tried, but without effect on the heart rate, and the patient was *in extremis*.

The medication and state of the heart can best be followed by daily notes:

November 15. Heart rapid (160 to 180) and regular all day.

November 16. 3 c.c. digalen given hypodermically during the day. Heart regular, 160 to 180.

November 17. *At midnight*, 0.5 mg. crystallin strophanthin given intramuscularly. 3 A.M., strophanthin repeated. 5.30 A.M., heart slow and irregular for two minutes. 6.30 to 9.30 A.M., heart slow and irregular, characteristic of auricular fibrillation (Fig. 2). 11.45 A.M., slow and irregular for two minutes. 12.30 P.M., 0.5 mg. crystallin strophanthin given. 10.20 P.M., heart slow and irregular for five minutes. 11 P.M., 0.5 mg., crystallin strophanthin given, a total of 2 mg. in twenty-four hours.

November 18. Continued rapid and regular until 10.30 A.M., when suddenly became slow (90 to 100) and perfectly regular. Great relief and improvement in patient's condition.

November 19. 6.30 A.M., again rapid, regular. 10.30 A.M., 0.5 mg. crystallin strophanthin given. 11 P.M., heart continues rapid, 160. 0.5 mg. crystallin strophthanthin given.

November 20. 10 A.M., rate 144. 1 mg. amorphous strophanthin given. 9 P.M., slow and very irregular for twenty minutes. In auricular fibrillation. 10.30 P.M., rate 164. 1 mg. amorphous strophanthin given.

November 21. 12.20 A.M. to 6.30 A.M., rate 88 to 96 and absolutely irregular. 9.30 A.M., rapid (150 to 160) and regular. 0.75 mg. amorphous strophanthin given. 10.30 A.M., to 1 P.M., slow (82 to 96) and perfectly regular. 7 P.M., rapid since 1 P.M. 1 mg. amorphous strophanthin given. 8.30 P.M., suddenly became slow (76) and perfectly regular. Tracings showed normal curves without evidence of block, alternation of coupling.

November 23. Continued slow and regular until 10.30 P.M., when suddenly became rapid.

November 24. 10.15 A.M., still rapid. 1 mg. amorphous strophanthin given.

11.30 A.M., slow and regular until 3.30 P.M., when became rapid. 9 P.M., rate 170. 1 mg. amorphous strophanthin given.

November 25. 8.30 A.M., continues rapid, regular. 1 mg. amorphous strophanthin given.

November 29. Heart has been slow and perfectly regular until today when it became rapid at 11.40 A.M. 2.30 P.M., 1 mg. amorphous strophanthin given. 3.40 P.M., paroxysm ceased.

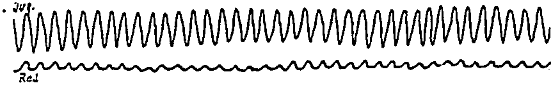

FIG. 1.—Tracing taken during a paroxysm. Pulse rate, 168.

During the next month the heart regained its compensation, though paroxysms recurred every three or four days. Many ceased in an hour or less, but if they lasted more than three hours 1 mg. of amorphous strophanthin was injected into a muscle and they invariably stopped within three hours and usually in about one hour. The rhythm between attacks was always normal.

FIG. 2.—Tracing taken November 17, 1911, between paroxysms of regular tachycardia. Rate, 110; auricular fibrillation.

From November 29, 1911, to January 20, 1912, strophanthin was administered on thirteen occasions. On the latter date tincture of digitalis was ordered and continued, with brief interruptions when there was evidence of poisoning. Thereafter, while there were many brief attacks, none lasted more than two hours until March 16, 1912, when a coryza developed with a temperature of 101°. A paroxysm began at 6 A.M. At 9 A.M. 1 mg. amorphous strophanthin was given and the attack ceased at 10 A.M., and the heart remained slow and regular all day. During the night the

FIG. 3.—Tracing taken after onset of permanent auricular fibrillation. Rate, 70; taking digitalis. Fig. 3 (original) could not be reproduced and was left out.

patient was very restless and had several short paroxysms. The heart again became rapid at 7 A.M., and at 10.30 A.M. showed some evidences of dilatation. Strophanthin was then given as usual, and the attack suddenly terminated in one hour.

He was not seen again until the next day, when he said that his heart had been irregular since the cessation of the last attack, and upon examination it showed the characteristics of auricular fibrillation (Fig. 3). This has continued to the present time, and no paroxysms of tachycardia have occurred. While the reserve accommodation force of the heart is slight and there is some pulmonary congestion and a small ascites, the patient is far more comfortable than before and is able to accomplish more work. Digitalis has constantly been necessary, and he is most comfortable when the heart rate is between 60 and 70, a point which I have observed in many instances of auricular fibrillation.

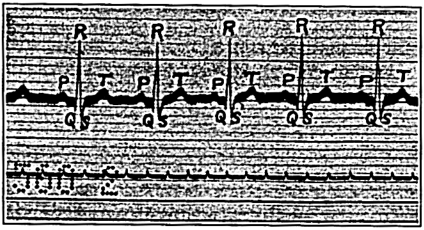

Fig. 4.—Normal electrocardiogram. *P* is due to auricular systole; *Q, R, S, T,* are due to ventricular systole; *P-Q* represents the time consumed in conducting the stimulus from the auricle to the ventricle; *T-P* represents the diastolic period. Time, 0.2 second.

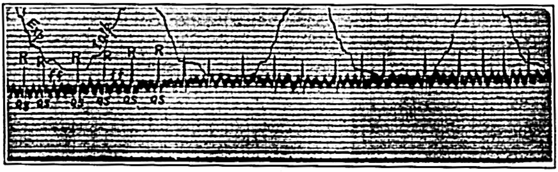

Fig. 5.—Patient's electrocardiogram. Lead II (right arm and left foot). Time, 0.2 second; pulse rate, 108.

It is unfortunate that no electrocardiograms were obtained from this patient during a paroxysm, but, despite repeated arrangements for them, the attacks never developed at a time and place where they could be taken. However, on April 2, 1912, two weeks after the onset of permanent auricular fibrillation, electrocardiographs records were taken by Dr. T. Stuart Hart, to whom I am greatly indebted for the ones here reproduced (Fig. 6) and for the interpretation which follows.

(NOTE. A normal electrocardiogram (Fig. 4) is shown to facilitate comparison with that from the case reported.)

The electrocardiograph records of this case (Fig. 5) show the usual features of auricular fibrillation:

1. Complete irregularity: the ventricular complexes are separated by unequal intervals; long and short diastolic periods follow one another haphazard without any rule or fixed relationship.
2. The *P* wave is absent.
3. There is a series of small waves (*ff*) which continue without interruption throughout the whole of the systole and diastole; these are probably due to the fibrillary activity of the auricle, and occur at a rate of about 375 per minute. The ventricular rate is 108 per minute.

This case presents certain special features. The ventricular complexes, of which the *R* waves are the most evident features, are distorted by the coincident occurrence of the fibrillation waves (*ff*). The *R* waves show a considerable variation in size and are influenced by two factors: they are largest at the end of expiration and smallest in the early part of inspiration, and they vary in height according as they occur coincident with a peak of a fibrillary wave or with a depression between the fibrillary waves. The waves *Q* and *S* are fairly well marked. The *T* waves are small, and for the most part so distorted by the simultaneous occurrence of the fibrillary waves that they can be made out only with great difficulty.

The waves of auricular activity (*ff*) are unusually large and fairly rhythmic. If one disregards the distortion caused by the simultaneous occurrence of the *R* waves of ventricular activity the rhythmic character of the fibrillary waves is seen extending over long periods. The rate of these waves is comparatively slow, for one often sees cases of fibrillation with a fibrillary rate between 500 and 600 per minute. These features—(1) the size, (2) the slowness, and (3) the rhythmicity—suggest that the mechanism of this case is one of auricular fibrillation, which is on the borderline not far removed from the cases of auricular flutter, in which are very rapid (up to 300) coördinated contractions of the auricles with a ventricle which responds to every second or third auricular impulse.

Lewis[1] has shown that the mechanism of auricular fibrillation, auricular flutter, and paroxysmal tachycardia is formation of impulses at an abnormal point (heterogenetic impulse formation) in the auricular wall. He concludes that in paroxysmal tachycardia the impulses are generated at a single point, while in auricular fibrillation impulses are generated incoördinately in many foci. In auricular flutter also there is, according to Ritchie,[2] "stimulation, excessive in frequency and intensity, of a localized area in some part of the auricular wall." It is, therefore, evident that, so long as the auricle is in fibrillation, paroxysms of regular tachycardia cannot occur. Hewlett[3] and Mackenzie[4] have also reported cases of paroxysmal tachycardia which under digitalis developed auricular fibrillation, sometimes temporary, with cessation of the attacks.

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[1] Mechanism of the Heart Beat, London, 1911, pp. 190–193; Heart, 1912–1913, iv, 171.

The only drugs of value in auricular fibrillation are the digitalis bodies, and it is in this condition that their best effects are seen. In auricular flutter, also, Lewis[5] and Ritchie[6] recommend digitalis, each reporting instances in which the normal rhythm has been restored by its use. The return to normal rhythm was often preceded by a short period of auricular fibrillation. Inasmuch as the mechanism of paroxysmal tachycardia is somewhat similar to that of auricular fibrillation, and almost exactly like that of auricular flutter, one might well expect benefit from the use of digitalis in regular tachycardia, such as was obtained by the administration of strophanthin in the case here reported.

The pharmacological action of strophanthin is the same as that of digitalis. It has many advantages in an urgent state, such as prolonged tachycardia with a dilating heart, notably ease of administration by intramuscular injection, which insures rapid absorption and, therefore, prompt effect.

Strophanthin is a powerful, and sometimes dangerous, drug, but it should often be repeated more frequently than is usually done, that is, until a satisfactory effect such as is obtained after several days of digitalis administration by mouth is secured. The patient must be watched carefully for indications of severe poisoning, especially a high grade of heart-block, which can be released by atropin. The induction of auricular fibrillation should not be feared, as, when that occurs, the tachycardia will cease, and it may be transitory, the heart subsequently returning to the normal rhythm. Even if it persists it is often a conservative state and reduces the limits of response very slightly.

I have had only one other opportunity to use strophanthin in prolonged and regular tachycardia as most attacks cease spontaneously or are relieved by vagal pressure. This was in a woman, aged forty-three years, who was brought to Bellevue Hospital with bronchopneumonia. She stated that during the previous four years she had suffered from frequent attacks of rapid heart action which began and ended abruptly. The present attack, which began shortly after the onset of the pneumonia, had lasted five days. The rate of the heart was about 200 per minute, and it was extremely dilated. Her feet were swollen and the liver was pulsating. She was given an intramuscular injection of 0.5 mg. crystallin strophanthin, and as no effect was noted this was repeated in three hours. Within a half-hour after the second injection auricular fibrillation developed and her distress was greatly relieved. On the next day the edema was lessened and her liver had ceased to pulsate. Auricular fibrillation continued until her death two days later from vasomotor failure and venous stasis did not recur. An autopsy was unobtainable.

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[2] Auricular Flutter, New York, 1914, p. 28.

[3] Jour. Amer. Med. Assoc., 1908, li, 655.

[4] Heart, 1910–1911, iii, 273.

[5] Loc. cit.

[6] Loc. cit.

Summary. A case of paroxysmal tachycardia with the ventricular form of the venous pulse is reported. Strophanthin was administered many times during the attacks and they always ceased following it,—usually in about one hour. On several occasions transitory fibrillation of the auricle terminated attacks. Finally under the combined effect of digitalis and strophanthin permanent auricular fibrillation was induced and no more paroxysms have occurred. The case illustrates the close relation between paroxysmal tachycardia, auricular fibrillation, and auricular flutter.

Strophanthin, in repeated doses if necessary, is recommended for prolonged attacks of paroxysmal tachycardia.

THE EFFECTS OF EXERCISE ON THE NORMAL AND PATHOLOGICAL HEART; BASED UPON THE STUDY OF ONE HUNDRED CASES.

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For a number of years the question of acute dilatation of the heart has been a debated one. Many attempts have been made to settle the question both by clinical observations and direct experiment, but these have led to no generally accepted conclusions. Until about a decade ago there was practically no difference of opinion on the subject. It was conceded that a normal heart when subjected to even relatively slight strains would dilate to a degree dependent upon the severity of the strain. If the heart were normal this dilatation was relatively soon recovered from—a sort of physiological dilatation. In the event of the heart already being weakened from disease this dilatation might become permanent.